

A Conceptual Model of the Spread of Rabies That Integrates Computer Simulation and Geographic Information Systems

Lorinda L Sheeler-Gordon,* Kenneth R Dixon

The Institute of Environmental and Human Health, Texas Tech University, Lubbock, TX

Abstract

Rabies, a viral infection of the central nervous system, is transmitted by direct contact with an infectious individual and is considered to be a predominately zoonotic disease. Epidemic models have focused on the spatial spread of rabies, and emphasized the importance of understanding the transmission and spread of the disease. This conceptual model concentrates on the geographic spread and transmission of rabies in raccoon populations. A stochastic, individual-based model that incorporates probabilities of contact between groups of the population will be integrated into a geographic information system (GIS) using the ARC/INFO macro language. We anticipate that the integration of computer simulation and GIS will assist in the development of an epidemic model predicting the geographic spread of rabies. The model was designed to investigate disease control scenarios, such as optimizing the placement of rabies oral vaccine to impede the further spread of disease.

Keywords: modeling, simulation, rabies, raccoon, epidemiology

Introduction

Rabies, a viral infection of the central nervous system, is transmitted by direct contact with an infectious individual and is considered to be predominantly a zoonotic disease. Rabies in wildlife such as bats, foxes, skunks, and raccoons occurs when the population of animals reaches a threshold density; hence, transmission is achieved by direct contact. If a human has contact with a rabid animal, rabies becomes a disease that affects humans. Although the incidence of rabies in humans is rare (with only a few deaths per year in the United States), it is a horrifying disease for which there is no known case of recovery after the onset of clinical symptoms (1).

The raccoon, *Procyon lotor*, is considered a major wildlife reservoir of rabies in the eastern United States and is currently spreading its distribution as a vector of the disease. In the 1950s, the first outbreak of raccoon rabies in the United States occurred in Florida, and the number of reported rabid raccoons is continuing to increase (2).

Raccoons are considered a solitary species. Home ranges of adult females overlap broadly and there is no evidence of territoriality, whereas home ranges of adult males overlap less than 10% with adjacent adult males. Males of neighboring home ranges were found to have a separation of at least 2 kilometers (km) (3,4). Barash (5) reports data on captured raccoons from neighboring areas and from widely separated areas. The interactions of the captured raccoons indicate some degree of neighbor recognition. The raccoons from widely separated areas exhibit hostile behavior toward each

* Lorinda L Sheeler-Gordon, Texas Tech University, The Institute of Environmental and Human Health, PO Box 41163, Lubbock, TX 79409-11 USA; (p) 806-885-4567; E-mail: lsheeler@ttu.edu

other, while neighboring individuals show tolerance for each other. These data provide evidence that males show territoriality only with other males (3,4,5). Adult males move around more than adult females, and males generally have a larger home range (3,4). Home range diameters have been reported as measuring 1 to 3 km, with suburban populations having smaller home ranges of 0.3 to 0.7 km (3). The home ranges of adult males and females overlap, but individuals usually remain apart by mutual avoidance, except during the mating period. The only groups having been reported together are family groups, communal winter dens, and those inhabiting areas of abundant food (3,6).

A low density of raccoons is considered to be 5 individuals per square kilometer (individuals/km²). High densities have been reported up to 20 individuals/km². In suburban areas, densities of as many as 68.7 individuals/km² have been reported. Ellis (7) radiotracked seven individual raccoons and concluded that when densities were high, raccoons seemed to move less and had smaller home ranges. In areas of high densities, raccoons were distributed evenly throughout all habitats. As densities decreased, however, the distribution favored particular habitats (4).

Raccoon long-range movements can be observed when juveniles disperse, or when environmental conditions and food availability are unfavorable. Reported data have much variability. Distances of 121 km (8), 266 km (9), and 254 km (10) have been recorded for adult raccoons. Butterfield (11) reported a maximum distance of 1.6 km, but with an average of 0.6 km. Distances for juvenile dispersal ranged from a few kilometers up to a maximum of 20 km.

Raccoons are classified as highly susceptible to rabies infection based on an intramuscular injection of the LD50 value (12). The LD50 for raccoon intraspecies transmission is 3.9 virus per inoculum (13). Because the virus concentration in saliva varies among individuals, the dosage transmitted when in contact with another individual varies, influencing the course of infection. An experimental inoculation of the LD50 value in a single dose by intramuscular injection, along with an exposure site of the neck or hind leg, is the best estimate of natural infection. It is noted that experimental inoculation cannot capture all the possibilities of natural exposure.

Exposure can induce the production of antibodies. Not all animals naturally exposed to rabies die of the disease. Field investigations of raccoons collected during an epizootic and observed for the following two years resulted in eight of ten raccoons surviving (14). Virus-neutralizing substances have been induced experimentally and have conferred resistance to further, massive inoculations of the rabies virus (13). Immunity or resistance to the rabies virus seems to play a role in naturally occurring raccoon populations.

Raccoons have been found during the day in residential areas and found with pet animals, resulting in increased human contact. Consequently, every year many people receive post-exposure treatment for exposure to wild animals. To minimize the number of human exposures and control and prevent the further spread of rabies, epidemic rabies models have focused on the spatial spread of the disease. Previous rabies models have focused on the fox as the main vector of the disease. The model presented here focuses on the raccoon as the main rabies vector.

The long incubation period of rabies in raccoons is a major factor in its transmission cycle. Therefore, animal movement was explored as a factor of transmission. The transmission cycle has two possible pathways. The first pathway is the migration of the

healthy raccoon that carries rabies to an uninfected geographic region. After the incubation period, the raccoon becomes infectious, experiences clinical symptoms, and spreads the disease to this new region. The second pathway is the abnormal behavior of the infectious raccoon whose confused movements result in the raccoon wandering and having contact with neighboring individuals. Both pathways are geographic in nature, and both transmission pathways are examined in this model. One objective of the model is to determine which mode of transmission has a larger role in the spread of rabies. A second objective is to assist in the development of rabies control strategies, thus reducing human exposures.

The Conceptual Model

A raccoon population in a rabies cycle can be divided into four categories or groups, depending on their disease status: susceptible raccoons; exposed raccoons (infected, but not infectious); immune raccoons; and infectious rabid raccoons (Figure 1). The susceptibles are those animals that previously have not been exposed to the virus or who have lost their immunity. A susceptible can only move into the exposed group. The exposed are those animals that have been exposed to an infectious, rabid animal. There is a variable incubation period of 39 to 79 days (13) during which rabies cannot be transmitted. The exposed animal can enter either the immune group or the infectious group. The category that the exposed animal moves to depends on the amount of virus to which the individual has been exposed. The immune group is composed of those animals that have been exposed to the rabies virus and have produced rabies-neutralizing antibodies. These animals are in this category for a variable length of time. Assuming that immunity in all animals is lost over time at the same rate, the rate of conversion from the immune group back to the susceptible group is treated the same for all animals that

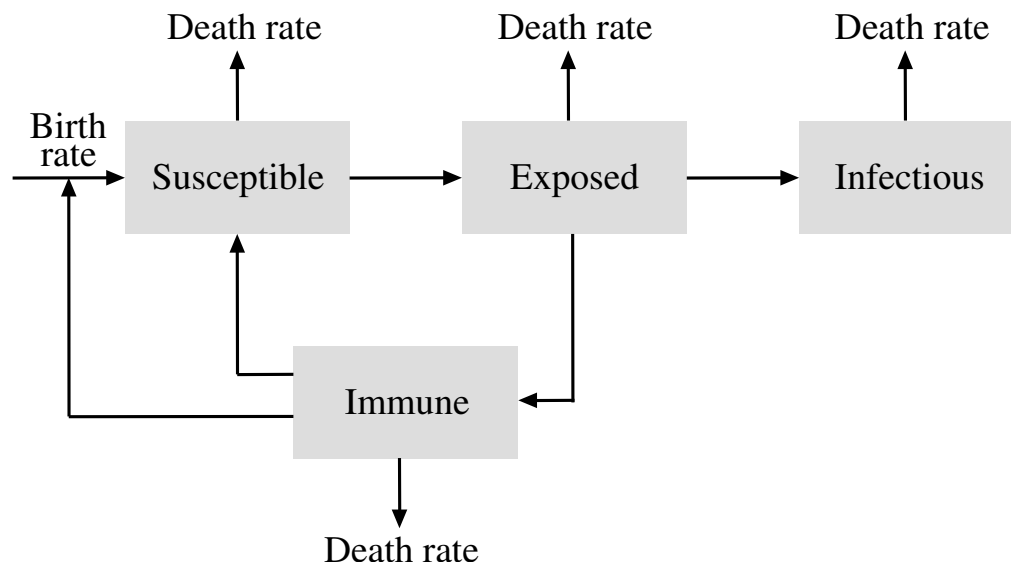


Figure 1 Epidemic model of the interactions between population processes (births and deaths) and disease processes among raccoon groups.

were exposed to an infectious animal. The infectious group is composed of those animals that show clinical symptoms for a short period of 3 to 8 days (13), and can transmit the disease by contact with the group of susceptibles. The infectious period ends in death, and the animal is removed from the population.

Young born to susceptibles and immunes are added to the susceptible group at a rate equal to the birth rate. Birth rate of the exposed group is assumed zero because of a gestation period of 63 days, and a weaning period of 2 to 4 months (4). Non-rabies death occurs in the susceptible, immune, and exposed groups, and is assumed to be the same rate for all groups. All individuals entering the infectious group are removed via the death rate, though death may occur from rabies or a non-rabies cause.

Infectious individuals having adequate contact with susceptible individuals results in a susceptible becoming an exposed individual. The probability of rabies transmission is the same as the contact rate. The amount of virus in the saliva is variable and is determined by a randomly drawn number from a lognormal frequency distribution produced from data on the prevalence of virus in saliva. The amount of virus and the LD50 value determine which category the exposed animals will enter. The dosage determines whether the individual will produce rabies-neutralizing antibodies or incubate the disease.

The incubation period in the exposed group is determined by a random variate drawn from a lognormal frequency distribution, which is produced from data of the known incubation period (15). After the incubation period, the exposed would then enter the infectious category. The length of stay in this category is also determined by a random variate drawn from a lognormal frequency distribution, which is produced from data of the known infectious period (15).

Conclusion

The long incubation period of rabies occurring in the exposed group is a major factor in the transmission cycle. The transmission cycle has two possible pathways, dispersal or home range. Using a geographic information system (GIS), a spatial grid was designed to simulate an extensive geographic area. The GIS allowed the data to be tracked over a period of time. The model uses the spatial procedures in ARC/INFO GIS, including GRID to simulate animal migration. The four status groups were tracked both spatially and temporally to analyze the pattern of the spread of the disease. The epidemic and animal movement models were integrated into the GIS using the ARC/INFO macro language (AML).

The animal movement model is a grid-based spatial model with grid cell size set equal to the average raccoon home range size (Figure 2). Raccoons are assigned to grid cells as individuals or as members of a social group. Individual raccoons then are assigned to one of the four status groups. For each individual in a given cell at time t , the model determines whether that individual moves to an adjacent cell (home range model), disperses to some more distant cell (dispersal model), or remains within the cell (Figure 3). The epidemic model then is used to determine the probability of individuals from the given cell infecting individuals in the cells contacted. Susceptible individuals have a probability of being infected by contact with those individuals that are infectious in contacted cells. After the interactions of individuals in each cell are determined, their group status is updated and the process repeats for each time step in the simulation.

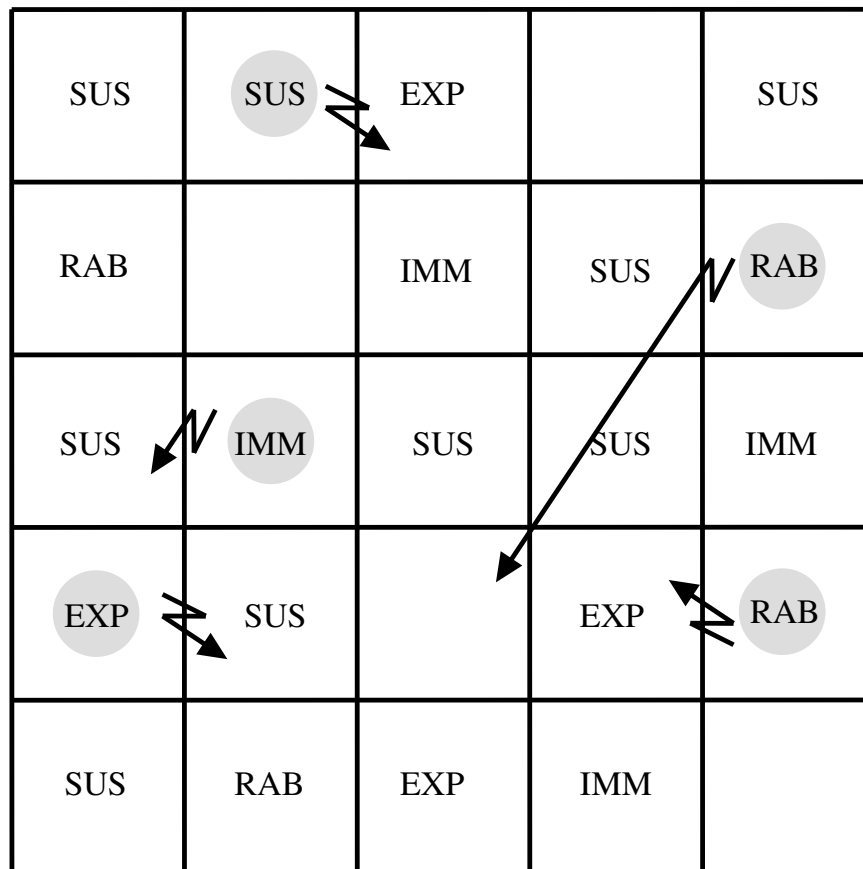


Figure 2 Movement of raccoons through a spatial grid. Each grid cell is equal to the size of the average home range. SUS = susceptible raccoon; EXP = exposed raccoon; IMM = immune raccoon; RAB = rabid infectious raccoon. Arrows represent movement to another grid cell.

Models of animal movement can be based upon any of the following three premises: the matching of spatial patterns of observed behavior (16), the set of rules arising from mechanisms governing the response of an individual to its environment (17), or theoretical constructs such as random walk models (18,19). The complete animal movement model incorporates features of all three methods within the home range. Models of dispersal distance are constrained random walk models. These models use transition probabilities that define the direction and distance moved, based on the animal's position relative to an activity center (20,21).

We anticipate that the integration of computer simulation and a GIS will assist in the development of an epidemic model that simulates the geographic spread of raccoon rabies. The model was designed to investigate disease control scenarios, such as optimizing the placement of rabies oral vaccine to impede the further spread of disease.

Obtaining adequate sample sizes for the estimation of the model's parameters will be essential for future research. Many hundreds of animals would have to be trapped to obtain reliable estimates of contact rates, concentrations of virus in saliva, antibody

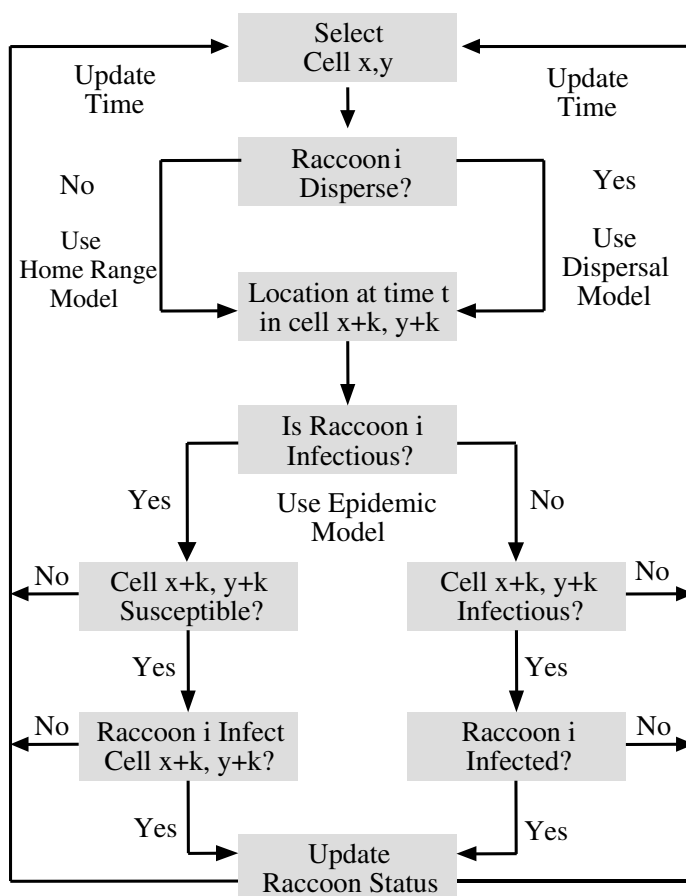


Figure 3 Flow diagram of contact among raccoon groups for the grid-based model.

prevalence, disease prevalence, and other factors. These estimates are necessary to achieve greater insight into the eventual control of wildlife rabies.

Acknowledgments

The authors wish to thank the Department of Biological Sciences and the Institute of Environmental and Human Health at Texas Tech University for financial support and use of facilities. We also thank the GIS in Public Health Conference planning committee for granting a student travel award, allowing attendance at the conference.

References

1. Murray JD, Stanley EA, Brown DL. 1986. On the spatial spread of rabies among foxes. *Proceedings of the Royal Society of London* B229:111–50.
2. Jenkins SR, Winkler WG. 1987. Descriptive epidemiology from an epizootic of raccoon rabies in the middle Atlantic states, 1982–1983. *American Journal of Epidemiology* 126:429–37.

3. Kaufmann JH. 1982. Raccoon and allies. In: *Wild mammals of North America: Biology, management, and economics*. Ed. JA Chapman, GA Feldhamer. Baltimore: The Johns Hopkins University Press. 567–85.
4. Lotze J, Anderson S. 1979. *Procyon lotor*. *Mammalian Species* 119:1–8.
5. Barash D. 1973. Neighbor recognition in two solitary carnivores: The raccoon and the red fox. *Science* 185:794–96.
6. Schwartz CW, Schwartz ER. 1981. *The wild mammals of Missouri*. Columbia, MO: University of Missouri Press and Missouri Department of Conservation.
7. Ellis RJ. 1964. Tracking raccoons by radio. *Journal of Wildlife Management* 28:363–68.
8. Giles LW. 1943. Evidences of raccoon mobility obtained by tagging. *Journal of Wildlife Management* 7:235.
9. Priedwert FW. 1961. Record of an extensive movement by a raccoon. *Journal of Mammalogy* 42:113.
10. Lynch GM. 1967. Long range movement of a raccoon in Manitoba. *Journal of Mammalogy* 48:659–60.
11. Butterfield RT. 1944. Populations, hunting pressure, and movement of Ohio raccoons. *Transactions of the North American Wildlife Conference* 9:337–43.
12. Kaplan C. 1985. Rabies: A worldwide disease. In: *Population dynamics of rabies in wildlife*. Ed. PJ Bacon. London: Academic Press. 1–22.
13. Carey AB. 1985. Multispecies rabies in the eastern United States. In: *Population dynamics of rabies in wildlife*. Ed. PJ Bacon. London: Academic Press. 23–42.
14. McLean RG. 1975. Raccoon rabies. In: *The natural history of rabies, vol. 2*. Ed. GM Baer. New York: Academic Press. 53–77.
15. Frerichs RR, Prawda J. 1975. A computer simulation model for the control of rabies in an urban area of Columbia. *Management Science* 22:411–21.
16. Siniff DB, Jensen CR. 1969. A simulation model of animal movement patterns. *Advances in Ecological Research* 6:185–219.
17. Wolff WF. 1994. An individual-oriented model of a wading bird nesting colony. *Ecological Modelling* 72:75–114.
18. Holgate P. 1971. Random walk models for animal behavior. In: *Statistical ecology, vol. 2*. Ed. GP Patil, EC Pielou, WC Waters. University Park, PA: Penn State University Press. 1–12.
19. Tyler JA, Rose KA. 1994. Individual variability and spatial heterogeneity in fish population models. *Reviews in Fish Biology and Fisheries* 4:91–123.
20. Dunn JE. 1978. Optimal sampling in radio telemetry studies of home range. In: *Time series and ecological processes*. Ed. HH Shugart. Philadelphia: SIAM Institute for Mathematics and Society. 53–70.
21. Dunn JE, Gipson PS. 1977. Analysis of radio telemetry data in studies of home range. *Biometrics* 33:85–101.